

# Initiation of ventricular fibrillation by supraventricular beats in patients with acute myocardial infarction

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**SUMMARY** The role of supraventricular extrasystoles in the initiation of ventricular arrhythmia was studied in 72 consecutive patients who developed primary ventricular fibrillation during the acute phase of myocardial infarction. In six patients (8%), a total of 12 episodes of ventricular fibrillation and 16 episodes of ventricular tachycardia were initiated by supraventricular extrasystoles. Ventricular fibrillation and tachycardia were initiated by single supraventricular extrasystoles in 16 and by salvos  $\geq$  two beats in 12 episodes. The RR coupling interval of the supraventricular impulse immediately preceding ventricular tachycardia ranged from 240 to 420 ms (mean 356 (62)) and was characteristic of R-on-T (prematurity index  $< 1$ ) in 63% of episodes. Average peak serum creatine kinase activity in the six patients in whom ventricular tachycardia was initiated by a supraventricular extrasystole was 1275 units compared with 720 units in the remaining 66 patients. Five of these six patients later showed evidence of pump failure. Lignocaine or procainamide or both suppressed the ventricular arrhythmia in five of the six patients.

The initiation of ventricular fibrillation or tachycardia by supraventricular extrasystoles in acute myocardial infarction is not uncommon and may reflect the increased vulnerability of the heart after a large infarct. These arrhythmias may respond to drugs that suppress ventricular irritability.

The initiation of ventricular arrhythmia by supraventricular extrasystoles is uncommon.<sup>1 2</sup> The initiation of ventricular fibrillation by supraventricular extrasystoles in patients with acute myocardial infarction has been rarely reported.<sup>3 4</sup> To assess the role of supraventricular extrasystoles in the initiation of ventricular fibrillation in patients with acute myocardial infarction we critically analysed the mode of initiation of the tachycardia in 72 consecutive patients in whom the onset of one or more episodes of ventricular fibrillation was recorded.

## Patients and methods

We studied 72 consecutive patients (61 men and 11 women, mean (SD) age 59(11)) admitted to the coronary care unit with acute myocardial infarction who developed primary ventricular fibrillation. All patients were admitted within 24 hours of the onset of chest pain, and the mean time taken to reach the coronary care unit was four hours. The diagnosis of acute myocardial infarction depended on all the following criteria being present: (a) a typical clinical history; (b) new Q waves or typical evolutionary ST-T changes; and (c) characteristic serial changes in the activity of serum creatine phosphokinase, alanine aminotransferase, and lactic dehydrogenase. The site of old and acute infarction was defined by the criteria of Lipman and Massie.<sup>5</sup> Primary ventricular fibrillation was defined as ventricular fibrillation in

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the absence of appreciable congestive failure, pulmonary oedema, and cardiogenic shock.<sup>6 7</sup>

In all 72 patients continuous electrocardiographic monitoring (4–108 hours per patient, mean (SD) 32 (28) hours) was performed. Each episode of non-sustained ventricular tachycardia (defined as  $\geq 3$  extrasystoles and lasting  $< 30$  seconds at a rate of  $\geq 100$  beats per minute), sustained ventricular tachycardia (lasting  $> 30$  seconds or requiring earlier termination because of haemodynamic compromise), and ventricular fibrillation was recorded, and the mechanism of initiation was analysed. The prematurity index of the supraventricular impulse that initiated ventricular tachycardia or ventricular fibrillation was determined by dividing its RR coupling interval by the QT interval of the basic supraventricular impulse. The basic QT interval was obtained by averaging the QT intervals of the 3–5 consecutive basic supraventricular impulses immediately preceding the one to which the supraventricular extrasystole was coupled. A prematurity index  $< 1$  was considered to represent short coupling or R-on-T phenomenon. Lignocaine infusion (1 to 4 mg/minute with an initial bolus injection of 75–100 mg) or procainamide infusion (2–6 mg/min after an initial dose of 1 mg/kg bodyweight over 10–20 minutes) or both were given to all patients who developed ventricular arrhythmia. None of these drugs was given before the onset of primary ventricular fibrillation.

## Results

A total of 152 episodes of primary ventricular fibrillation were recorded in the 72 patients. Twelve episodes (8%) in six (8%) patients were initiated by supraventricular impulses. In addition, eight runs of non-sustained ventricular tachycardia and eight episodes of sustained ventricular tachycardia were also initiated by supraventricular impulses in the same six patients. None of the patients had evidence of accessory atrioventricular pathways. The basic heart rate preceding the onset of ventricular arrhythmia in

the six patients was not significantly different from that of the rest of the group. The frequency of supraventricular extrasystoles in these six patients and the remaining patients could not be compared because data were not available for all 66. Nevertheless, paroxysmal supraventricular arrhythmias (supraventricular tachycardia and atrial flutter and fibrillation) occurred in three (50%) of the six patients and in 15% of the rest of the group.

Table 1 summarises the clinical data in the six patients. Five patients had acute anterior wall myocardial infarction. Two patients developed right bundle branch block and left anterior hemiblock and a third patient developed isolated left anterior hemiblock. A mean peak serum creatine kinase activity of 1275 U/l (compared with 729 U/l in the remaining 66 patients) and an adverse clinical outcome suggested extensive myocardial damage in these six patients. Five of the six patients developed congestive heart failure, two patients died during initial hospital stay from pump failure, and a third patient died after a prolonged complicated course after rupture of the interventricular septum. All the episodes of ventricular fibrillation that we analysed occurred in the absence of pronounced congestive failure, pulmonary oedema, and cardiogenic shock and thus conformed to the criteria of primary ventricular fibrillation. One patient was admitted with a clinical picture consistent with intermittent coronary artery spasm and went on to develop extensive anterior wall myocardial infarction within 24 hours. Three episodes of non-sustained ventricular tachycardia and one episode of ventricular fibrillation initiated by supraventricular extrasystoles occurred during periods of ST elevation associated with ST-T alternans (fig 1).

Table 2 summarises the mode of initiation of 12 episodes of primary ventricular fibrillation and 16 episodes of non-sustained or sustained ventricular tachycardia by supraventricular impulses. The tachycardia was initiated by a single supraventricular extrasystole in 16 episodes, by two supraventricular extrasystoles in nine episodes, and by  $\geq 3$  extrasystoles in three. The QRS complex of

Table 1 Clinical data in six patients with primary ventricular fibrillation initiated by supraventricular extrasystoles

Case	Site of infarction	Peak CK	BBB	Follow up
1	Anterior	1725	RBBB + LAH	Patient died from late pump failure
2	Anterior	1275		Discharged from hospital
3	Anterior	1215	RBBB + LAH	Patient died from late pump failure
4	Anterior + old inferior	1425	LAH	Patient developed VSD and later died from pump failure
5	Anterior	900		Discharged from hospital
6	Inferior + old anterior	740		Discharged from hospital

CK, serum creatine kinase; BBB, bundle branch block; RBBB, right bundle branch block; LAH, left anterior hemiblock; VSD, ventricular septal defect.

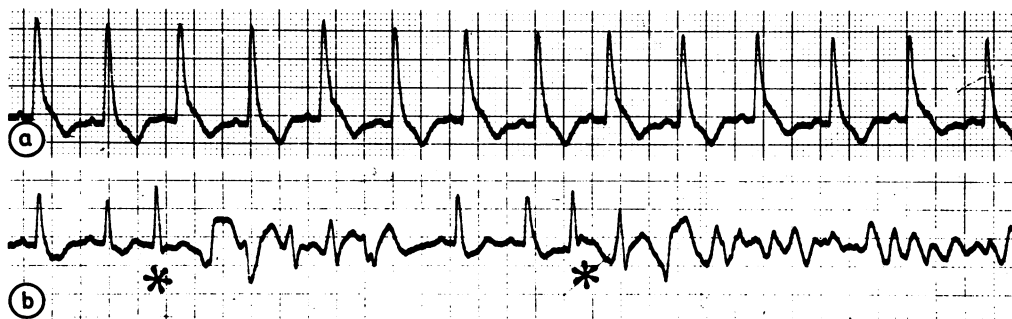


Fig 1 Electrocardiogram showing non-sustained ventricular tachycardia and ventricular fibrillation initiated by supraventricular extrasystoles (asterisks) in a patient with Prinzmetal angina associated with ST-T alternans. Extensive anterior wall myocardial infarction developed within 24 hours of onset of symptoms.

the supraventricular extrasystole initiating ventricular arrhythmia was similar to the QRS complex of the basic supraventricular rhythm. In the 16 episodes in which the tachycardia was initiated by a single supraventricular extrasystole the RR coupling interval of the extrasystole ranged from 240 to 420 ms (mean (SD) 356 (62) ms) and had a prematurity index  $<1$  (R-on-T phenomenon) in 10 (63%) of 16 episodes. There was no significant difference between the prematurity index of the supraventricular impulse that initiated an episode of ventricular tachycardia or ventricular fibrillation in the same patient. In some patients the coupling interval of the supraventricular extrasystole initiating non-sustained ventricular tachycardia was shorter than the one initiating ventricular fibrillation (fig 2), while in other patients the reverse was true. There was also no significant difference in the basic heart rate preceding the onset of either type of tachycardia in the same patient. In five of the six patients at least one episode of ventricular tachycardia or ventricular fibrillation was also initiated by ventricular extrasystoles. The coupling interval of the ventricular extrasystole that initiated the tachycardia was not significantly different from the RR interval of the supraventricular extrasystole that induced the arrhythmia in the same patient. In the remaining patient all episodes of ventricular arrhythmia were initiated by supraventricular impulses (fig 3). Panels a and b of fig 3 show two episodes of sustained ventricular tachycardia each initiated by a single supraventricular extrasystole. Panel c shows the onset of ventricular fibrillation after two closely coupled supraventricular extrasystoles, while panel d shows ventricular fibrillation after an episode of supraventricular tachycardia.

All episodes of ventricular fibrillation and some

episodes of ventricular tachycardia required cardioversion. In the six patients in whom ventricular arrhythmia was initiated by supraventricular extrasystoles, the arrhythmia was suppressed by lignocaine in two patients. The frequency of supraventricular extrasystoles before and after suppression of the ventricular arrhythmia was evaluated in those two patients and showed no appreciable change. After lignocaine or procainamide or both, ventricular fibrillation did not recur in three other patients but non-sustained ventricular tachycardia was only partially suppressed. In the sixth patient bretylium infusion and overdrive ventricular pacing were required to suppress the tachycardia.

## Discussion

The present report shows that initiation of primary ventricular fibrillation by supraventricular extrasystoles in acute myocardial infarction is uncommon but by no means rare. We use the term supraventricular extrasystoles rather than atrial extrasystoles because, in the absence of intracardiac

Table 2 Mode of initiation of ventricular arrhythmias by supraventricular impulses

Mode of initiation	Number of episodes		
	NSVT	SVT	VF
Single SVES	5	3	8
Two SVES	3	3	3
SVT	0	2	1
Total	8	8	12

NSVT, non-sustained ventricular tachycardia; SVT, sustained ventricular tachycardia; VF, ventricular fibrillation; SVES, supraventricular extrasystole; SVT, supraventricular tachycardia defined as  $\geq 3$  at the rate of  $\geq 100$  beats/min.

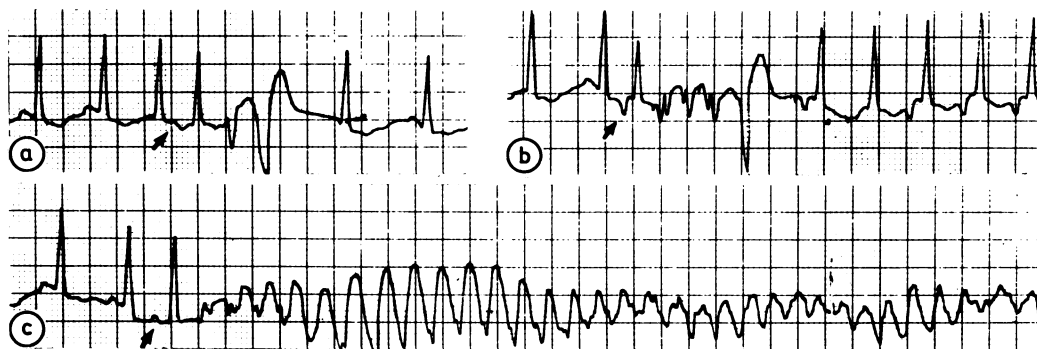


Fig 2 Electrocardiograms showing supraventricular extrasystoles (arrows) initiating ventricular couplets (a), non-sustained ventricular tachycardia (b), and ventricular fibrillation (c) in a patient with acute myocardial infarction.

recordings, atrioventricular junctional beats could not be excluded. In 8% of the patients who developed primary ventricular fibrillation and in 8% of the episodes of ventricular fibrillation, the arrhythmia was initiated by supraventricular impulses. In those patients, several episodes of non-sustained or

sustained ventricular tachycardia were also initiated by supraventricular impulses. The prematurity index of the supraventricular QRS complex initiating 63% of the episodes of ventricular tachycardia was  $<1$ , and was thus characteristic of an R-on-T phenomenon. This was explained by an

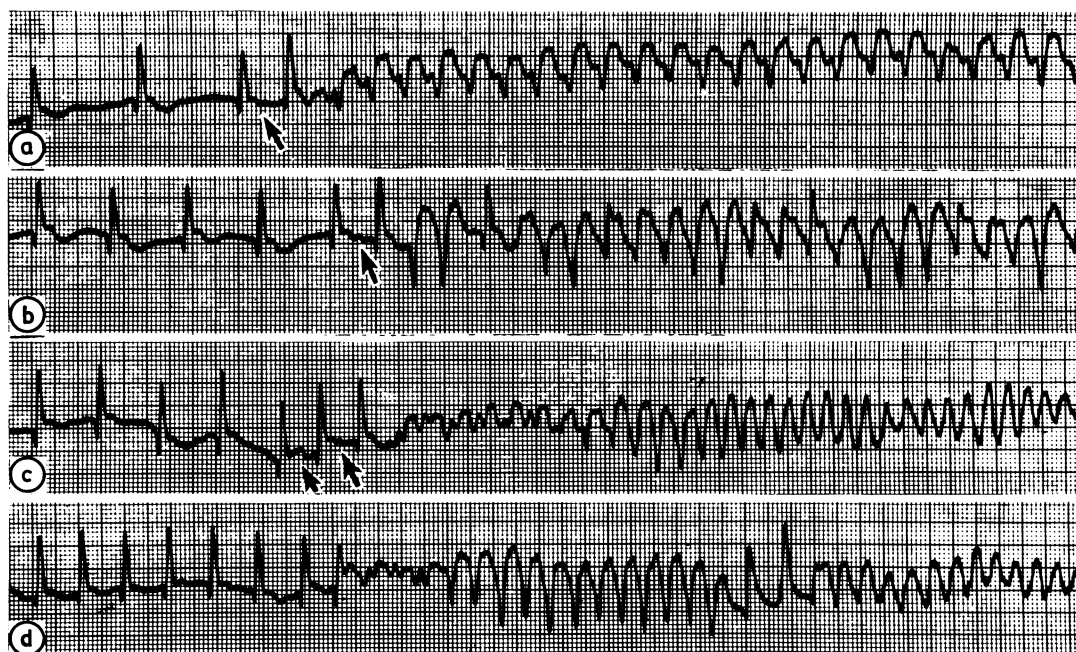


Fig 3 Electrocardiograms obtained from a patient with acute myocardial infarction showing (a) the initiation of uniform ventricular tachycardia by a supraventricular extrasystole, (b) the initiation of multiform ventricular tachycardia by a supraventricular extrasystole, (c) the initiation of ventricular fibrillation by two supraventricular extrasystoles, and (d) the initiation of ventricular fibrillation after supraventricular tachycardia. Supraventricular extrasystoles are indicated by arrows.

early coupling of the premature P wave to the preceding QRS complex in the presence of a normal or only slightly prolonged PR interval. This may suggest a certain degree of enhanced atrioventricular conduction in some of those patients probably secondary to increased catecholamine concentrations. In the remaining 37% of episodes, however, the coupling interval of the supraventricular QRS complex that initiated the arrhythmia was long. This is not significantly different from the previously reported frequency of 41% of episodes of primary ventricular fibrillation initiated by ventricular extrasystoles with a prematurity index of  $> 1$ .<sup>3</sup> Also there was no significant difference in the heart rate preceding arrhythmias initiated by ventricular or supraventricular extrasystoles. The only recognisable difference between the small group of patients in whom primary ventricular fibrillation was initiated by supraventricular extrasystoles and the larger group in whom the arrhythmia was prompted by ventricular extrasystoles was that the former group seemed to have a larger infarct and more extensive myocardial damage. This was evident from a higher peak creatine kinase activity and a more complicated course, with congestive heart failure in five of the six patients and death from terminal pump failure in three of them. It is reasonable to speculate that the more extensive myocardial ischaemia may have increased ventricular vulnerability.

Under experimental conditions it is not uncommon to induce ventricular fibrillation by supraventricular extrasystoles if these are properly timed and if ventricular vulnerability is sufficiently altered. For example, ventricular fibrillation was induced by supraventricular extrasystoles in infant goats and pigs because earlier coupling could be achieved since in these animals the refractory period of the atrioventricular conduction system is shorter than that of the ventricle.<sup>8</sup> On the other hand, very early coupling of supraventricular beats is not required to induce ventricular fibrillation if ventricular vulnerability is altered by experimental acute ischaemia,<sup>9</sup> subacute ischaemia,<sup>10</sup> or hypothermia.<sup>11</sup> These interventions can lengthen the refractory period, and if combined with a critical degree of spatial non-uniform distribution (characterised by adjacent regions of short and long refractoriness) could create "ventricular vulnerability"—in other words, the necessary electrophysiological conditions for extrasystoles to initiate reentrant excitation.<sup>12</sup>

The concept of ventricular vulnerability was first described in animal models by Wiggers and Wegria who showed that ventricular arrhythmias could be induced by a single test stimulus applied during the relative refractory period.<sup>13</sup> This work introduced

the concept of a triggering mechanism—(that is the critically coupled extrasystole) and an appropriate electrophysiological precondition (that is non-uniform refractoriness during the vulnerable period), setting the stage for reentrant excitation. Later the clinical expression of this concept was highlighted by the phrase "R-on-T phenomenon".<sup>14 15</sup> There are at least two limitations to extrapolating from these initial experimental observations to spontaneous ventricular arrhythmias in the clinical setting. First, although in the normal heart the vulnerable period is limited to the QT interval, ischaemia can extend the vulnerable phase throughout a major portion of the diastolic interval,<sup>10</sup> so that ventricular arrhythmias caused by reentry could be initiated by a ventricular extrasystole falling outside the QT interval. The present study shows that the same is true when the triggering mechanism of the ventricular arrhythmia is a supraventricular extrasystole. Secondly, the concept of a triggering stimulus and an appropriate electrophysiological basis for arrhythmia can only be applied to the situation when the ventricular arrhythmia is induced in the electrophysiological laboratory by critically coupled ventricular or supraventricular electrical stimuli, and when, as in the present study, the arrhythmia is initiated by spontaneous supraventricular extrasystoles. On the other hand, when a spontaneous ventricular arrhythmia is triggered by a ventricular extrasystole two different mechanisms could be invoked. One, that the first ventricular extrasystole represents the discharge of an automatic focus that triggers a subsequent arrhythmia based on reentry. The other possibility is that both the initiating and subsequent impulses are part and parcel of the same electrophysiological mechanism of reentrant excitation. The two electrophysiological mechanisms have been clearly demonstrated in the canine post-infarction model<sup>16</sup> but cannot be discerned in the clinical setting. In at least two of our six patients the ventricular arrhythmia was apparently suppressed by drugs that may have altered the underlying electrophysiological conditions for reentry without affecting the supraventricular triggering mechanism.

We conclude that initiation of ventricular fibrillation by supraventricular extrasystoles in acute myocardial infarction is not uncommon. The presence of extensive myocardial injury may result in a greater degree of altered ventricular vulnerability and provides the necessary electrophysiological basis for the initiation of the ventricular arrhythmia by supraventricular extrasystoles. These arrhythmias may respond to drugs that suppress ventricular irritability without affecting the supraventricular triggering mechanism.

## References

- 1 Zipes DP, Foster PR, Tromp PJ, Pedersen DH. Atrial induction of ventricular tachycardia: reentry versus triggered automaticity. *Am J Cardiol* 1979;44:1-8.
- 2 Wellens HJJ, Bar FW, Farre J, Ross DL, Weiner I, Vonagt EJ. Initiation and termination of ventricular tachycardia by supraventricular stimuli. Incidence and electrophysiologic determinants as observed during programmed stimulation of the heart. *Am J Cardiol* 1980;46:576-82.
- 3 El-Sherif N, Myerburg RJ, Scherlag BJ, et al. Electrocardiographic antecedents of primary ventricular fibrillation. *Br Heart J* 1976;38:415-22.
- 4 Myerburg RJ, Sung RJ, Gerstenblith G, Mallon SM, Castellanos A Jr. Ventricular ectopic activity after premature atrial beats in acute myocardial infarction. *Br Heart J* 1977;39:1033-7.
- 5 Lipman BS, Massie E. *Clinical scalar electrocardiography*. 5th ed. Chicago: Year Book Medical Publishers, 1965.
- 6 Meltzer LE, Kitchell JR. The incidence of arrhythmias associated with acute myocardial infarction. *Prog Cardiovasc Dis* 1966;9:50-63.
- 7 Lawrie DM, Greenwood TW, Goddard M, et al. A coronary-care unit in the routine management of acute myocardial infarction. *Lancet* 1967;ii:109-14.
- 8 Preston JB, McFadden S, Moe GK. Atrioventricular transmission in young mammals. *Am J Physiol* 1959;197:236-40.
- 9 Carroll SE, Ahuja SP, Manning GW. The initiation of ventricular tachycardia and fibrillation in experimental coronary artery occlusion. *Am J Cardiol* 1965;16:813-9.
- 10 El-Sherif N, Hope LR, Scherlag BJ, Lazzara R. Reentrant ventricular arrhythmias in the late myocardial infarction period. 2. Patterns of initiation and termination of reentry. *Circulation* 1977;55:702-19.
- 11 Wallace AG, Mignone RJ. Physiologic evidence concerning the re-entry hypothesis for ectopic beats. *Am Heart J* 1966;72:60-70.
- 12 Gough WB, Mehra R, Restivo M, Zeiler RH, El-Sherif N. Reentrant ventricular arrhythmias in the late myocardial infarction period in the dog. 13. Correlation of activation and refractory maps. *Circ Res* 1985;57:432-42.
- 13 Wiggers CJ, Wegria R. Ventricular fibrillation due to single, localized induction and condenser shocks applied during the vulnerable phase of ventricular systole. *Am J Physiol* 1940;128:500-5.
- 14 Smirk FH. R waves interrupting T waves. *Br Heart J* 1949;11:23-9.
- 15 Smirk FH, Palmer DG. A myocardial syndrome: with particular reference to the occurrence of sudden death and of premature systoles interrupting antecedent T waves. *Am J Cardiol* 1960;6:620-9.
- 16 El-Sherif N, Gough WB, Zeiler RH, Hariman R. Reentrant ventricular arrhythmias in the late myocardial infarction period. 12. Spontaneous versus induced reentry and intramural versus epicardial circuits. *J Am Coll Cardiol* 1985;6:124-32.